Pollution and People

Asbestos fibres and the environment

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Large differences in cancer risk appear in the different epidemiological studies on asbestos workers. For mesotheliomas especially the type of fibre seems to be important, as we saw in the last article (p 551)—crocidolite and amosite being much more hazardous than chrysotile. This is supported by electron microscopy studies of postmortem specimens. In a series of mesothelioma cases from North America the tumours were associated with high fibre counts of amosite and crocidolite, but equal amounts of chrysotile were found in cases and controls.[1] Chrysotile fibres, however, have the magnesium leached out of them and are more soluble than the others, though a silicate skeleton may remain.[2] Thus the final proportions of fibres in the lungs may not be representative of the actual exposures.[3] Carcinogenesis probably requires persistence of the fibres but this is not proved.

Influence of fibre size and shape

There are other striking differences in the epidemiological data. One is that mesotheliomas seem to be caused by Cape but not Transvaal crocidolite.[2] Another is the difference in risk of lung cancer—and indeed of total mortality—between different groups of workers. The observed: expected ratio (in 13 studies of workers first exposed in the dusty conditions before 1945 or even earlier) ranges from 1·2:1 in some of the chrysotile miners and millers to 17:1 in insulation and textile workers exposed to a mixture of fibres.[4] Apart from different conditions of work—including whether the asbestos is wet or dry and dusty—the physical form of the fibre is important.[2] In chrysotile mining, for example, though it used to be very dusty, the fibres tend not to be properly separated and are thus in the form of bundles of relatively large diameter, with particles clinging to them. The finer the fibre to which workers are exposed the more readily it will remain airborne and be inhaled—and penetrate deeply into the lungs. In general crocidolite (fig) is the finest fibre, but that from the Transvaal has three times the diameter—and so nine times the falling speed—of Cape crocidolite, being similar to amosite. Chrysotile is also very fine but is curly and more likely to be intercepted before it penetrates far into the lung or reaches the pleura, though it can split into fibrils. Length is relevant too since very short fibres can be removed by phagocytes and very long ones will be limited in their movements.

Thus fibre size and shape are important factors in carcinogenesis—perhaps more so than chemical composition, especially for mesotheliomas. This is also suggested by experimental work. In inhalation studies in rats chrysotile, though eliminated from the lungs much more readily than crocidolite, appeared to be at least as carcinogenic as chrysotile or more so.[5,6] This was attributed to the use of finer chrysotile than had been customary in industry, and in one experiment[6] to a higher proportion of fibres over 20 μm in the chrysotile. In such experiments, however, the fibres may be ingested as well as inhaled and they are not complete replicas of human exposure. A cell culture study, correlated with animal studies, on several types of fibrous (and non-fibrous) mineral dusts pointed to a possible way of predicting risk: the most successful tests predicted a risk of mesotheliomas and fibrosis from fibres of 10 μm or more long and 1·4 μm or less in diameter.[7]

This may mean that the tendency towards finer processing of chrysotile should be discouraged,[8] though not all authorities believe that this follows from the experimental evidence. Equally the findings may be relevant to other types of natural fibrous material and to man-made fibres. Glass and mineral wool fibres are generally much coarser and the evidence suggests that they are safer than asbestos;[9] but a large-scale evaluation is in progress. Surprising incidences of mesothelioma in general populations (chiefly in rural Turkey) have recently been discovered in association with local fibrous mineral deposits[10]—notably with a fine, fibrous zeolite, used for building,[11] and with a material containing the amphibole asbestos tremolite (with a very small percentage of chrysotile), used for stucco and whitewash.[12]

Measuring asbestos

Asbestos concentrations are normally expressed in numbers of fibres per unit of tissue, air, or fluid, or in nanograms (electron microscopy). The current British hygiene standards specify limits of 0·2 fibres/ml for airborne crocidolite, 0·5 fibres/ml for amosite, and 1 fibre/ml for chrysotile. Large fibres and asbestos bodies, which form more readily on long amphibole than on chrysotile fibres in the tissues, can be counted satisfactorily with optical microscopy; but the finer and smaller fibres can be detected only by electron microscopy. The ratio of electron microscopy to optical microscopy values is not, however, constant. Though electron microscopy gives more accurate results it cannot be used routinely, being time-consuming and highly skilled work.

Asbestos in the general environment

The general public may be exposed to asbestos in several ways. "True" environmental exposure to asbestos should be distinguished from "paraoccupational" (in the homes of workers) and "neighbourhood" (near industry, mines, etc), and also from leisure-time exposure (working with asbestos products).[13] These last three aspects, notes an EEC report, are "areas of concern."[13] If the stricter controls introduced and recommended in recent years[14] are properly applied there should in fact be little need for concern, but clearly there has to

British Medical Journal, London WC1H 9JR
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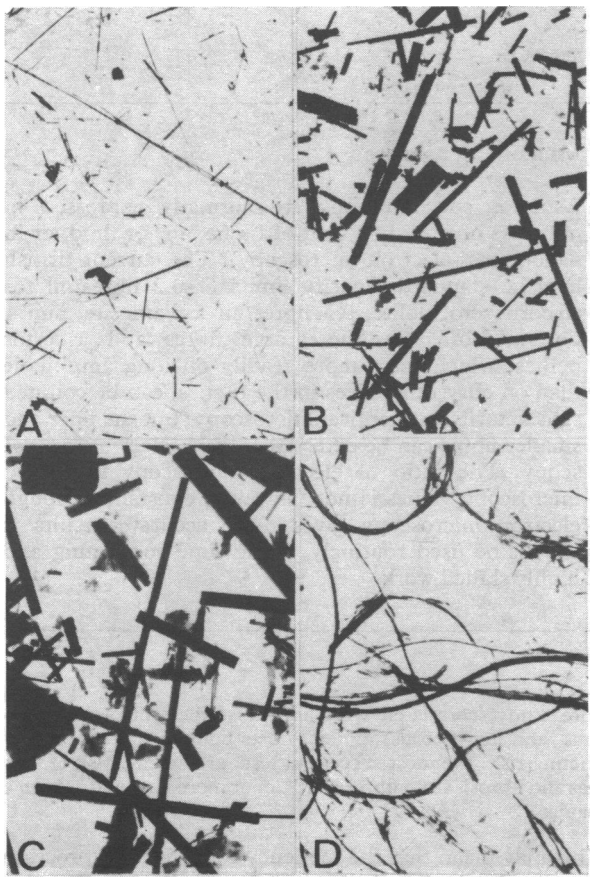
be great vigilance. True environmental exposure is mainly through the ambient air, water, beverages, and food. The two chief scientific issues are finding out the concentrations of fibres in different environments and looking for evidence of increased rates of cancer in populations thought to be specially exposed.

An American study looked at the general cancer mortality in United States counties in relation to asbestos deposits but found no correlation.[15] Clearly, however, any small effect could be masked by many factors, and more specific studies are more likely to be informative. The relation of asbestos in water to cancer rates has been the subject of several investigations.



Electron micrographs of crocidolite (A), amosite (B), anthophyllite (C), and chrysotile (D). Reproduced from Timbrell[2] by courtesy of the International Agency for Research on Cancer.

ASBESTOS IN WATER AND FOOD

Food and drink are contaminated with asbestos for various reasons. Many water supplies contain fibres, because of asbestos deposits (rare in Britain) in the area they come from or industrial pollution, or because fibres are leached from asbestos cement pipes. Accounting for perhaps a third of all water pipes in Britain, these have been used chiefly since 1945; the use of crocidolite ceased in the late 1960s. Preliminary studies have suggested that water supplies in Britain are not highly contaminated.[16] Asbestos is also widely used in the food industry in filters and in processing equipment. For products that cannot be heat treated, such as beer, wine, and soft drinks, the use of chrysotile in filters is "unsurpassed," according to the 1979 report on asbestos of the Scientific Committee for Food of the Commission of the European Communities. Such filtration removes bacterial and other contaminants and also, paradoxically, may reduce the number of asbestos fibres in the final product. Nevertheless, appreciable fibre counts have been found in a wide range of beverages as well as water samples.[17]

The EEC report[13] quotes a calculation suggesting that someone drinking 2 litres a day of the most contaminated water for 60 years would ingest no more than 0·07 g of asbestos, compared with an estimated 2-336 g for exposed workers—in whom an extra risk of gastrointestinal cancer has not always been found. According to another calculation, a man of average weight would have to drink some 10 million litres of the most contaminated water in his lifetime to ingest a quantity of asbestos corresponding to the doses that have produced tumours in animals, whereas no one is likely to drink more than 100 000 litres.[13] Moreover, the proportion of short, possibly less hazardous fibres is much higher in water and other general environments.[13] Even so, cancer incidences must be compared in different exposed populations.

In a Connecticut study no correlation appeared between gastrointestinal cancer and asbestos cement pipes (giving rise to short fibres) in the public water supply—the age and the length of the pipes and ability of the water to leach out the fibres being taken into account.[18] A continuing investigation is in progress in Minnesota because the water supplies of Duluth became more heavily contaminated with amphibole asbestos (again in the form of short fibres) when the dumping of mining waste in Lake Superior began in 1955. Comparison of gastrointestinal cancer rates in Duluth and two control cities showed no striking trend, though any final conclusion would be premature.[19] In Quebec, towns with the more contaminated drinking water had an excess of cancers of the lung and stomach in men and of the pancreas in women, but as these were asbestos mining towns occupational exposure could well have been responsible.[20] Recently significantly raised incidences of certain tumours have been reported in the San Francisco Bay region in the areas with the higher concentrations of chrysotile (from natural sources) in drinking water.[21] Associations were strongest with cancer of the lung in men, gall bladder and pancreas in women, and peritoneum in both sexes, but were also significant for stomach, oesophageal, renal, and pleural cancers; and the authors point out that cancers of the stomach in men and pancreas in women were actually increased in the two previous studies. Though occupation and socioeconomic factors were allowed for in the San Franscisco data, the authors themselves say that a case-control study is needed with information on such things as smoking and drinking habits. Without this clearly the findings do not permit definite conclusions.

The balance of the evidence from different types of work does not point to a risk from ingested asbestos in the general population. If there is no threshold of risk for gastrointestinal cancer associated with asbestos—a point that is regarded as uncertain—it has been suggested that since large populations receive their water through asbestos cement pipes some extra cancers would be expected, however low the risk.[8] But some authorities consider that even this is to overstate the probabilities.

ASBESTOS IN THE ATMOSPHERE

Asbestos fibres are widespread in the atmosphere (even on the Yorkshire moors[14]) and have many origins—natural and industrial sources and buildings, for example.[22] There are relatively few precise data.[13 22] Optical microscopy is not adequate for the small fibres, mixed with many other types of particles, found in the ambient air, and electron microscopy is required.[22] In general, concentrations are probably of the order of 1000 times lower than in industry under current standards,[13] though they may be much higher near industrial sites[13] and a considerable range of chrysotile concentrations is found in some studies.[22] Furthermore, the importance of such small fibres is questionable.

Asbestos fibres appear to be shed in greatest quantity from insulation board, followed by sprayed asbestos, asbestos cement sheeting, and other asbestos-containing products (in that order).[13] Over 60% of the asbestos imported into Britain is used in building construction.[14] Until the late 1960s sprayed asbestos, including crocidolite, was widely used; and there has been extensive lagging of pipes in public buildings, factories, etc, with asbestos that often contains a mixture of chrysotile and crocidolite or amosite. Although neither sprayed

asbestos nor crocidolite is now used (and in insulation products asbestos has largely been replaced), much remains in buildings. This has to be either sealed or in some cases removed, with careful inspection of the buildings concerned, which some but not all local authorities now carry out. The large-scale use of often accessible sprayed asbestos in schools in the United States, where unacceptable air concentrations have been found, poses an expensive problem.[23 24] School activities—and vandalism—are likely to make fibres more readily released.

When high concentrations have been reported in buildings in Britain the source has usually been asbestos that had been sprayed on and not sealed, especially if the activities in the building have produced appreciable disturbance of the air; some readings have shown levels exceeding the occupational hygiene standard for crocidolite even where there was little or no damage to the asbestos.[14] In general, however, the fibre concentrations in buildings with some components containing asbestos are very low. In a preliminary survey (carried out by the Health and Safety Executive and the Department of the Environment) in several different buildings using electron microscopy, the highest reading for airborne crocidolite was under 0·2% of the occupational standard. Nevertheless, authorities must be vigilant for the worst possibilities—excessive wear and tear, home improvement jobs, children playing, vandalism—and inspection and prompt action are clearly important. For this reason some of the cases mentioned in *Asbestos—Killer Dust*[25] are worrying.

The safe disposal of asbestos waste is also important[4 14]: dumps must not be accessible to the public, nor be later dug up or exposed for any reason. Hazards from natural fibrous deposits must equally be watched for.[10] Recently a Californian recreational area with exposed chrysotile turned out to give motorcyclists dust exposures approaching industrial limits.[26]

Environmental asbestos and public policy

The extreme view has been put forward that asbestos can never be safe and that all uses should be banned.[25] (Sir Richard Doll does not, as has been claimed, hold this view—he stated that crocidolite should be banned but was misreported.[27]) This would be neither practicable nor desirable at present, when adequate (or fully evaluated) substitutes are not available for all uses. Some people, including the Society for the Prevention of Asbestosis and Industrial Diseases, hold that asbestos should be restricted to a few essential uses. This may be achieved in time; some countries, such as Sweden and Denmark, are moving towards it now. The change will be less disruptive if it proceeds gradually—and this is happening. Legislation now being considered in Britain (for example, banning spraying and the use of asbestos in new insulation products) will largely formalise voluntary controls. The main public health issue concerns the asbestos we already have. How worried should we be about this?

Although, in general, fibre concentrations in the general atmosphere seem to be of the order of 1000 times less than in industry today, the population at large cannot be considered in the same way as those who are occupationally exposed. In the general environment there is no control over what happens to the asbestos as there is in industry. Moreover, the general public is likely to contain more vulnerable people than industry[13]—the opposite of the "healthy worker effect"—though whether they will be more vulnerable to the effects of asbestos is not clear. There has also been some suggestion, from an occupational study, that those exposed at younger ages had a higher cancer risk[28]; but whether this would apply to environmental exposure is not known. Finally, environmental exposure in many cases is continuous and for a lifetime. For all these reasons, plentiful safety margins must be allowed for the general population.[13] In addition, industrial hygiene standards were set to protect against incipient asbestosis rather than cancer (and it is too soon to say what risk, if any, of the various cancers remains under current standards). The EEC report concludes that air concentrations in the region of 1000 times lower than in industry do represent a sufficient safety margin. But it emphasises the need to keep levels as low as possible throughout the environment, with the help of good industrial practices and attention to the effects of asbestos-containing products in everyday life.

Those who campaign against asbestos argue that mesotheliomas may be caused by minimal contact with any fibre type.[29] Mesotheliomas have been associated with brief—but intensive—exposure to crocidolite and probably amosite (see last article, p 551), but for the rest the argument depends on anecdotal cases. These are regarded as unreliable evidence since mesotheliomas are thought to be sometimes "spontaneous"—or the people concerned may in reality have been heavily exposed in one way or another. Even though we may suspect a no-threshold dose-response relationship, such reports, it has been said, have to be set against the epidemiological evidence from the dusty industries of the past: in one factory, for instance, using chrysotile and a small proportion of crocidolite, only one man out of about 15 000 exposed for less than two years since 1933 is known to have developed a mesothelioma.[30] No one knows, it has been pointed out, how many council estates have tenants unaware that they should not be drilling, sanding, and tampering with the asbestos in their houses.[31] Clearly people should be aware that they have asbestos and that they must treat it with respect; but to spread alarm about a "'cancer risk' for council tenants" (as one headline put it) seems totally unjustified.

I am grateful for helpful discussion with Dr P C Elmes, Dr J C Wagner, and other members of the MRC Pneumoconiosis Unit, Llandough Hospital, Penarth; Dr J C Gilson, formerly director of the MRC Pneumoconiosis Unit; Dr Muriel L Newhouse, TUC Centenary Institute of Occupational Health, London School of Hygiene and Tropical Medicine; Mr W Penney, Asbestos Information Centre, London; and Mrs Nancy Tait, Society for the Prevention of Asbestosis and Industrial Diseases.

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Lesson of the Week

Double pathology as a cause of occult gastrointestinal blood loss

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The routine use of gastrointestinal endoscopy in most large medical centres has allowed rapid evaluation of the upper gastrointestinal tract in patients presenting with gastrointestinal blood loss, either acute or chronic. Endoscopic examination of the colon is of no value in massive bleeding of the lower gastrointestinal tract because of the difficulty in cleaning the colon,[1] but colonoscopy is invaluable in cases of chronic blood loss attributed to the large bowel.[2 3]

Small and clinically silent lesions are frequently shown with endoscopy and in the absence of other pathology are considered to be the cause of blood loss. We report on two patients with chronic gastrointestinal blood loss in whom small lesions not bleeding at the time of endoscopic examination were seen in the stomach and thought to be the cause of blood loss. In both patients further investigations showed lesions of the colon that certainly contributed to the blood loss. Care must be taken before accepting that small lesions seen on endoscopy cause chronic gastrointestinal bleeding.

Small lesions seen on endoscopy in one part of the gastrointestinal tract may not be the only source of chronic blood loss

Case reports

Case 1—A 56-year-old woman presented with fatigue. She was anaemic with a haemoglobin concentration of 8·6 g/dl and a microcytic blood film. Serum iron studies showed marked desaturation. She had no symptoms referable to the upper gastrointestinal tract and no recent change in bowel habit. The only drug medication was compound aspirin (consisting of 1200 mg/day of acetylsalicylic acid) for osteoarthritis of the hands. There were no abnormalities on examination. Rectal examination was normal but occult blood testing of the stools was positive on three occasions. Endoscopic examination of the upper gastrointestinal tract showed patchy antral gastritis which was more severe in the prepyloric region. The mucosa was friable and bled on contact. Biopsy specimens of the gastritic mucosa showed acute inflammation. She was transfused and treated with oral cimetidine 1 g/day for one month. Oral iron was also prescribed. Her haemoglobin concentration on discharge was 11·6 g/dl. Paracetamol was substituted for the aspirin. Three months later her haemoglobin concentration was 8·9 g/dl and she again had symptoms of anaemia. Stools were again positive for occult blood. Repeat endoscopic examination of the upper gastrointestinal tract showed minimal changes of antral gastritis with no bleeding on contact. Biopsy specimens showed changes of mild chronic inflammation and a marked reduction in the acute inflammatory cell infiltrate compared with the previous specimens. Examination with double-contrast barium enema showed a polypoid tumour 3 cm in diameter in the mid-transverse colon. A hemicolectomy was performed, and the tumour was a moderately well differentiated adenocarcinoma (Dukes's grade II). Nine months after surgery she was well. Her haemoglobin concentration has been steady at 12·2 g/dl.

Case 2—A 68-year-old man was first seen in 1977, when he presented with symptoms of anaemia. Physical examination showed pallor but no other abnormalities. Rectal examination was normal and stools were negative for occult blood. His haemoglobin concentration was 9·3 g/dl and mean cell volume was 79 fl (μm^3). Endoscopic examination of the upper gastrointestinal tract showed two small pyloric canal ulcers. Biopsy specimens showed acute inflammation. He was treated with antacids. He remained well and was seen three years later

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